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How Does SARS-CoV-2 Fragment the QRS?

LETTER TO THE EDITOR

To the Editor,

We eagerly read the article by Özdemir et al[1] about a retrospective study of the prognostic impact of QRS fragmentation (f-QRS) in 201 patients with severe COVID-19. It was found that the presence of f-QRS on electrocardiogram (ECG) is associated with increased in-hospital all-cause mortality in patients with severe COVID-19 as compared to those without f-QRS.[1] The study is appealing but raises concerns that require discussion.

The main shortcoming is that neither in patients with f-QRS nor in those without f-QRS in-hospital ECGs were compared with those recorded prior to admission. As long as pre-existing f-QRS was not properly excluded, a causal relation between SARS-CoV-2 and f-QRS cannot be established. Assuming that f-QRS was present already prior to the infection, it is conceivable that the overall mortality was higher because patients with pre-morbid cardiac compromise generally have a worse outcome from COVID-19 as compared to those who are cardiologically healthy.

Missing is a comparison between ECG findings and echocardiographic data. It has to be told if those with f-QRS also had more frequent systolic dysfunction, dyskinesia, hypokinesia, akinesia, myocardial thickening, or Takotsubo syndrome[2] than those without.

Missing is a comparison between ECG findings and long-term ECG results. It has to be told if those with f-QRS had more frequently supra- or ventricular ectopic beats, supra- or ventricular arrhythmias, or QT-prolongation compared to those without f-QRS.

Since cardiac involvement in SARS-CoV-2 infections can manifest with endocarditis, myocarditis, or pericarditis, it has to be known how many of those with f-QRS had endocardial, myocardial, or pericardial involvement in the SARS-CoV-2 infection. Endocarditis needs to be excluded by trans-esophageal echocardiography or biopsy. Myocarditis needs to be excluded by cardiac MRI with gadolinium or by endo-myocardial biopsy, and pericarditis needs to be documented by pericardial puncture.

Missing are the autopsy data. There is an urgent need to compare ECG findings with autopsy data as the golden standard for diagnosing any type and degree of cardiac involvement in the viral infection.

Missing is the repetition of the ECG recordings to see how reproducible the results were and if f-QRS resolved with recovery from COVID-19. Since the study had a retrospective design, it is conceivable that at least in some cases f-QRS resulted from poor quality of ECG recordings, thus representing an artifact.

There is also no subclass analysis of those who had undergone coronary angiography prior to the infection. Thus, it is recommended to re-evaluate the data with regard to previous coronary heart disease. It should be known how many of those with f-QRS had a previous myocardial infarction.

Additionally, investigations on the influence of the autonomic cardiac innervation on QRS fragmentation are missing. Since Guillain-Barre syndrome is a frequent

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complication of SARS-CoV-2 infections[3] and since GBS can go along with autonomic dysfunction,[4] it is crucial to know how many of those with f-QRS in fact had developed GBS with autonomic impairment.

Overall, the study has several limitations which challenge the results and their interpretation. Before attributing f-QRS to a SARS-CoV-2 infection, all differentials need to be thoroughly excluded. Correlation of f-QRS with morphological and functional cardiac parameters is warranted.

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